about the *true* paternity of the pregnancy that was affected with eclampsia. They were interviewed either antepartum, postpartum, or at a postnatal visit, but always after confidentiality had been established. The questioning was either direct or oblique, depending on my assessment of the patient's personality. The placenta was examined after delivery in all cases to exclude gross placental abnormalities such as hydatidiform mole coexisting with a normal fetus. To assess the prevalence of change of paternity in this area two random groups of 300 and 200 pregnant multiparous patients were questioned in 1971 and 1977 respectively.

Thirty-four (74%) out of the 46 multiparous eclamptic patients seen in my practice during the 10 years admitted that they had had a new partner for the affected pregnancy. Four were not sure, and eight were certain that there was no new factor in the affected pregnancy, which varied between their 3rd and 8th. In 20 cases the fact of a new partner was easily elicited during routine history taking, and subsequent interview was unnecessary. These new partnerships were owing either to death of the previous husband, separation, divorce and remarriage, unfaithfulness, or unmarried multiparous patients (Nnachi custom practised by the Igbo people). So far 14 of these patients have had subsequent deliveries under my supervision. Three had oedema and albuminuria but none had a recurrence of eclampsia. Two of them reported another change of partner.

Of the random groups of 300 and 200 women questioned in 1971 and 1977, respectively, for change of paternity, 31 and 12 had had changes in the preceding three years. Out of these 43, one had had intrapartum eclampsia and three had had only albuminuria and ankle oedema in their pregnancies.

Comment

The cause of gestosis remains enigmatic but may have an immunological basis.¹ The disease is associated with many and profound changes in the maternal state.² One view widely held is that primigravida patients are more commonly affected than multigravida. In Nigeria we certainly see multiparous patients with eclampsia. In this study of 46 multiparous eclamptic patients with singleton pregnancies 34 had had new partners for their pregnancies. The pregnancies in these multiparous patients became complicated by eclampsia, which is otherwise commoner in primigravida patients. For each particular new couple, however, the pregnancy was primary, irrespective of the gravidity of the woman. Whatever the aetiological factors, the manifestational sequence of gestosis seems the reverse of rhesus sensitisation, in which the chances of being affected increases with the number of pregnancies. This study suggests that when a multiparous patient with a singleton pregnancy develops eclampsia it is likely to be a primary pregnancy.

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¹ Scott JS, Jones WR, eds. *Immunology of human reproduction*. London and New York: Academic Press, 1976.

² Leading article. Immunological factor in pre-eclampsia. *Br Med J* 1976; ii:604-5.

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Mittelschmerz is a preovulatory symptom

Many women experience mid-cycle lower abdominal pain (mittelschmerz) during ovulatory cycles. It may be caused by intraperitoneal release of follicular contents at ovulation or by muscular cramps in the uterus, tubes, or large bowel,¹ but the exact relation of mittelschmerz to ovulation has not been defined. We investigated its timing as part of a study of the periovulatory period using ultrasonic techniques.

Patients, methods, and results

A group of 96 women were studied. All had regular ovulatory menstrual cycles and were attending the reproductive biology clinic of the Royal Women's Hospital. From the mid-follicular phase of the cycle (day 9-11) onwards blood was drawn daily to measure luteinising hormone (LH)

concentration by rapid radioimmunoassay, and each patient was questioned about her symptoms during the preceding 24 hours. The diameter of the developing follicle was measured in three planes and a mean calculated using either a static B-scanner (Diasonograph N E 4200) or a real-time device (A D R Model 2130).² This procedure was continued daily until the follicular appearances of ovulation² were first noted.

Thirty-four women (35%) noticed lower abdominal pain, usually lasting six to 12 hours, during the mid-cycle period. In 27 discomfort was localised to one or other iliac fossa, which in all but two cases corresponded to the

Relationship of mid-cycle pain to the peak plasma LH concentration (day 0) in 34 women

	<Day -2	Day -1	Day 0 (LH peak)	Day +1
Timing of pain relative to peak LH concentration	2	2	25	5*

*Four of these patients did not ovulate on ultrasound evidence until day +2.

side of the developing follicle. In seven patients the pain was central and suprapubic. Most patients (77%) felt pain on the day of the peak plasma LH concentration (table). When the data were analysed in relation to the ultrasonically determined time of ovulation it was found that in 31 (91%) the pain had occurred in the previous 24-48 hours. The mean ($\pm$ SD) follicular diameter on the day of pain was 19.3 ± 2.2 mm. Ovulation was confirmed in every cycle by a mid-luteal 24-hour urinary pregnanediol content of greater than 2.0 mg,³ and four of the cycles were conceptual. Pain did not last longer than 24 hours in any patient and none required analgesia.

Comment

In all but one patient, whose pain might have coincided with follicular rupture, the intact follicle could still be seen after the pain had disappeared. As in previous studies,² the plasma LH peak concentration preceded "ultrasonic ovulation" by about 24 hours. In most cases mittelschmerz coincided with the plasma LH peak—that is, when the follicle was still enlarging. Nevertheless, simple follicular distension is unlikely to be the cause of pain since even on the day of peak volume many follicles were distorted by distension of the bladder, suggesting a low intrafollicular pressure. Furthermore, in anovulatory cycles cystic follicles may become much bigger without producing pain. In mammals intrafollicular pressures are low and no significant preovulatory pressure differentials have been noted.⁴ On the other hand, the mid-cycle LH rise induces increased contractility in ovarian perfollicular smooth muscle,⁵ an effect which is partially mediated through production of prostaglandin $F_{2\alpha}$. This may be the origin of mittelschmerz.

The recognition of mittelschmerz as a preovulatory event has useful implications. It may help to identify the most fertile day of mid-cycle, and thus help the timing of coitus or artificial insemination, and may also improve the efficacy of natural family planning techniques.

¹ Jeffcoate TNA. *Principles of gynaecology*, 4th ed. London: Butterworth, 1975:544.

² Hackeloe BJ, Fleming R, Robinson HP, Adam AH, Coutts JR. Correlation of ultrasonic and endocrinologic assessment of human follicular development. *Am J Obstet Gynec* 1979;135:122-8.

³ Pepperell RJ, Brown JB, Evans JH, Rennie GL, Burger H. Investigation of ovarian function by measurement of urinary oestrogen and pregnanediol excretion. *Br J Obstet Gynaec* 1975;80:321-33.

⁴ Espey LL, Lipner H. Measurements of intrafollicular pressures in the rabbit ovary. *Am J Physiol* 1963;205:1067-72.

⁵ Wright KH, Wallach EE, Fromm E, Jeutter DC. Studies of rabbit ovarian contractility using chronically implanted transducers. *Fertil Steril* 1976;27:310-8.

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